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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

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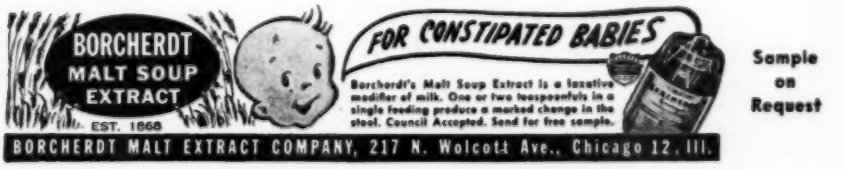
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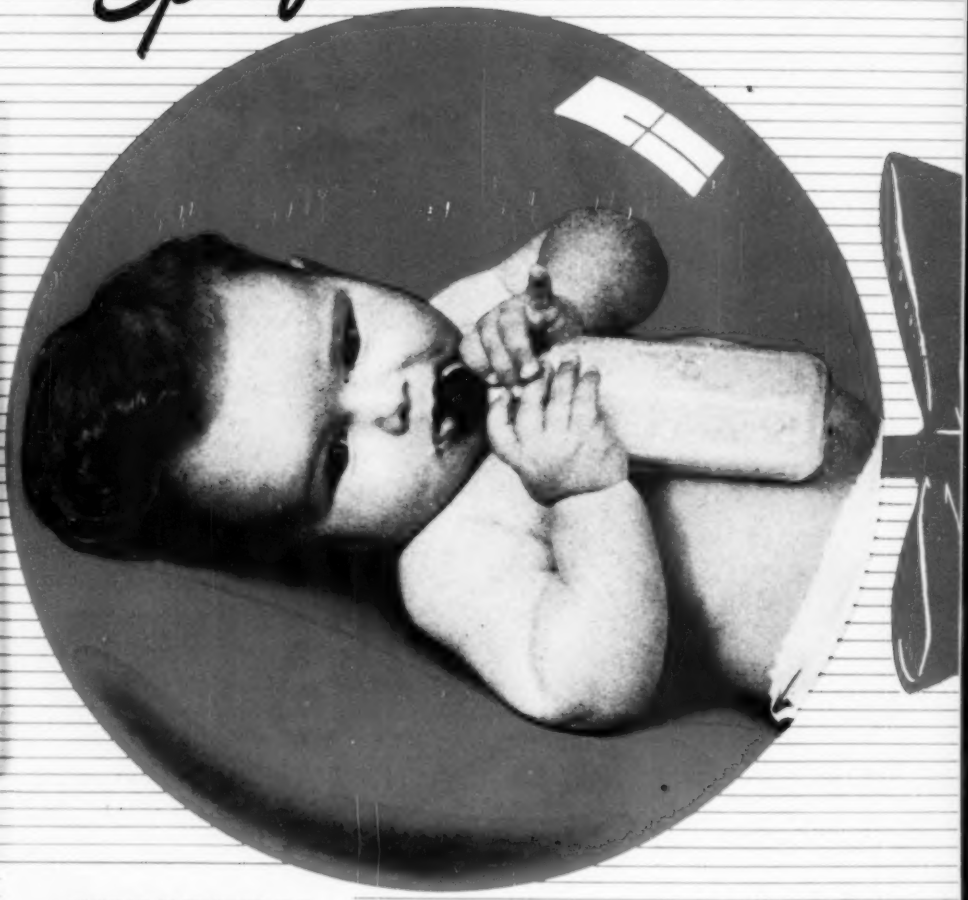

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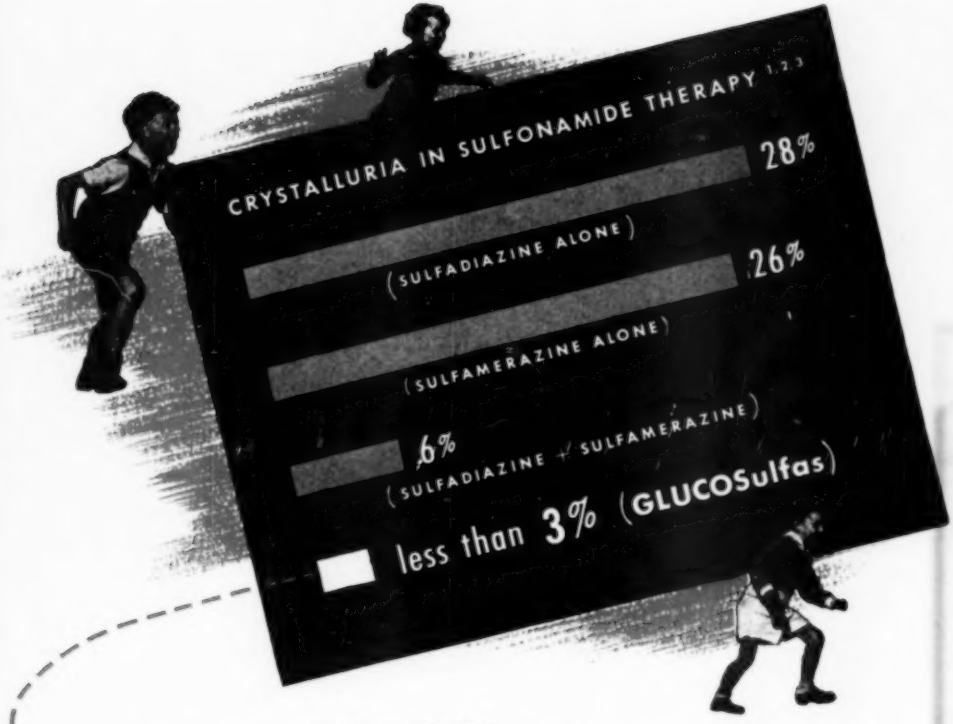

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1. Flippin, H. F., and Reinhold, J. G.: Ann. Int. Med. 25:433, 1946.
2. Ledbetter, J. H., and Cronheim, G. E.: Am. J. Med. Sci. 216:27, 1948.
3. Frisk, A. R., et al: Brit. Med. J. 1:7, 1947.

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✻ 1. Cannon, A. Benson, and McRae, Marvin E.: Treatment of Scabies, J.A.M.A. *138*:557 (Oct. 23) 1948.

2. Wooldridge, W. E.: The Gamma Isomer of Hexachlorocyclohexane in the Treatment of Scabies. J. Invest. Dermat. *10*:363 (May) 1948.

3. Niedelman, M.L.: Treatment of Common Skin Diseases in Infants and Children. J. Pediat. *32*:566 (May) 1948.

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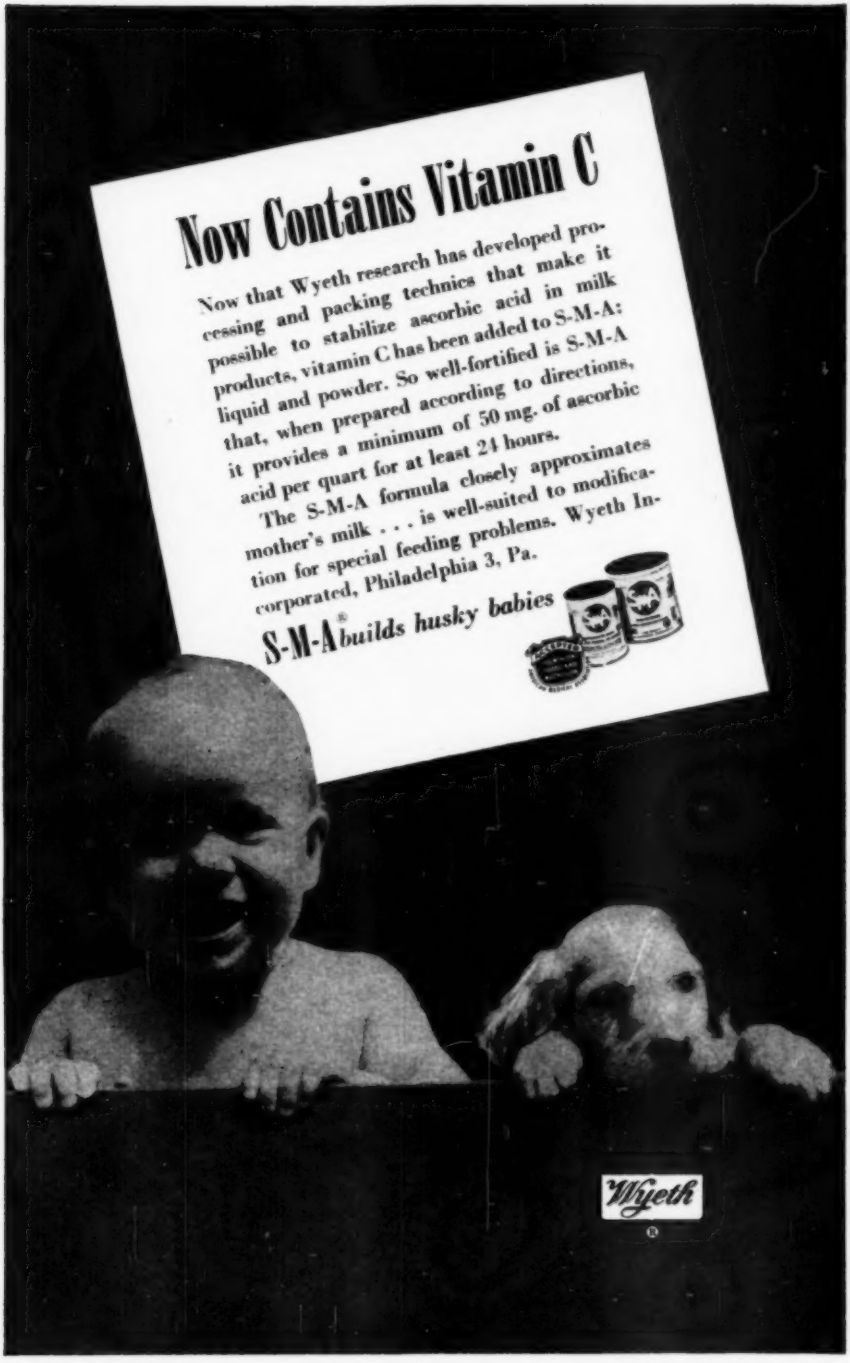
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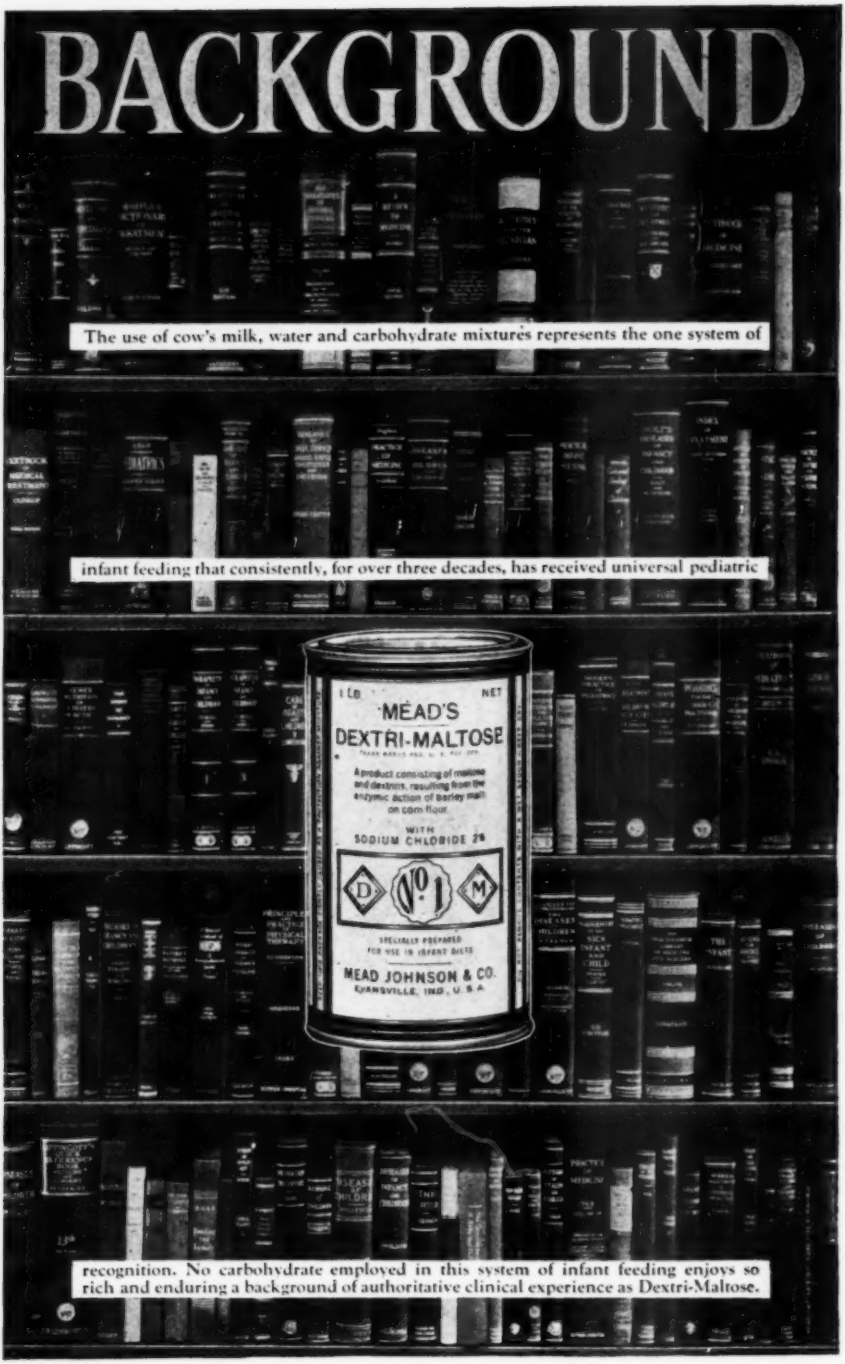
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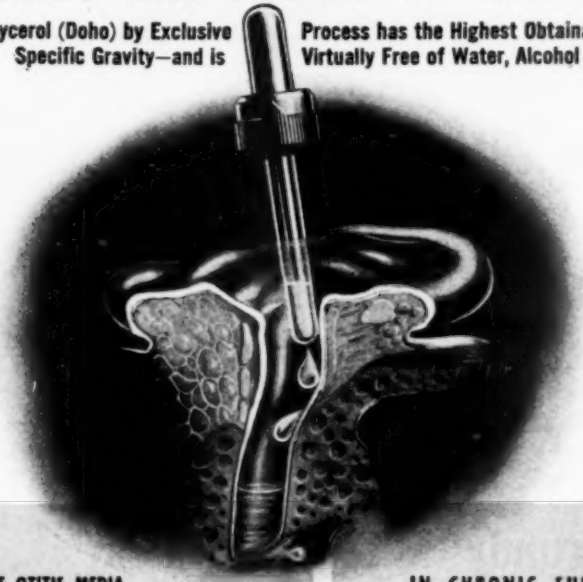

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OBSTRUCTION OF THE BILE DUCTS, HYPOGLYCEMIA AND ATELECTASIS*

REPORT OF CASE

HARRY R. LITCHFIELD, M.D. F.A.C.P.

AND

ARMAND H. MOSS, M.D.

Brooklyn.

Obstruction of the biliary system is not infrequently encountered in pediatric practice. This report represents a study and discussion of such a case seen in the pediatric wards of the Beth-El Hospital.

CASE REPORT

A young mother, non-diabetic, without any previous history of illness, was delivered on April 25, 1947 of her first baby at term without difficulty The newborn male (G) weighed six pounds four ounces.

From the start the baby had a stormy course. Soon after delivery the infant was found to be extremely cyanotic, with a weak cry and cold extremities. There were some râles over both bases, anteriorly and posteriorly. Heart sounds were apparently normal. An immediate blood sugar was advised. The report showed there were 10 mg. per cent sugar in 100 cc. of blood.

The infant was put into a plastic oxygen tent. A continuous intravenous of 10 per cent glucose in saline was given; penicillin, 10,000 units was administered every three hours from April 25 to April 28. Hykinone, ampule 2.4 mg., 1 every four hours, was

*From the Pediatric Service, Beth-El Hospital, Brooklyn, New York.

injected intramuscularly on April 25 as a precautionary measure for four days.

In spite of all these procedures, the condition of the infant remained poor, and on several occasions the infant had to be resuscitated by artificial respiration, administration of oxygen under high concentration and one injection of two minims of adrenalin subcutaneously. Cyanosis was constant and was only controlled by continuous oxygen. The respirations continued to be shallow, and signs of poor aeration in both lung fields persisted for one week. The persistent cyanosis was suggestive of a congenital heart lesion. This was corroborated by a large shadow of the heart in the x-ray film. The x-ray findings also revealed bilateral congenital atelectasis. An electrocardiogram, taken on May 5, was suggestive of a congenital cardiac lesion; another on May 7 revealed sinus tachycardia, right axis deviation, and a third on May 24 was suggestive of auricular tachycardia.

The clinical picture was confused by the presence of 150 erythroblasts per 100 white cells in the blood smear, although father and mother and baby were all Rh positive. Mother and baby were Group O.

On the fifth day, a collected specimen of urine was dark amber in color, and positive for bile. On the eighth day, the jaundice in the infant became more pronounced. This was to be an outstanding sign throughout his stay in the hospital.

At the end of one week's stay in the nursery, the infant was transferred to the pediatric ward for further study.

Examination of the Infant. The baby was in a plastic oxygen tent, the respirations were noted to be rapid and shallow. The anterior fontanel was slightly sunken. Breath sounds over both lungs were faint. Percussion of the heart showed it to be moderately enlarged, no murmurs were heard. Abdomen was soft; liver and spleen were not enlarged. The infant weighed six pounds four ounces. A specimen of urine was dark yellow, strongly bile stained. The stools, collected for twenty-four hours, were clay colored with some mucus and tinged with blood. Blood examination revealed hemoglobin 119 per cent, red blood cells 4,500,000, white blood cells 6,100. There were still 44 erythroblasts per 100 white blood cells.

Progress Notes. During the remaining weeks in the hospital,

the picture of cyanosis and respiratory difficulties gradually gave place to increasing jaundice of the skin and sclera. There were times when it was impossible to remove the infant from the plastic oxygen-hood because of the marked cyanosis which developed soon after the infant was out of the tent. However, within a week the infant improved while out of the oxygen tent. Clinically and radiologically the lungs showed expansion, except for small areas in the right upper lung field. The blood smears were free of erythroblasts.

On May 6 the icterus index was 86. Bile and urobilinogen were absent from the feces. Urobilinogen was present in the urine in 1:10 dilution. Bile in urine was positive. Cephalin-flocculation test was two plus in eighteen hours; the van den Bergh positive direct immediate. The fragility of the red cells remained within normal limits: on June 2, 1947, normal, began 0.44, complete 0.32; control, began 0.42, complete 0.34. Prothrombin time was somewhat prolonged. May 10, 1947, quick 17 sec.; normal 12 sec. Other prothrombins taken during hospitalization: June 2, 1947, quick 22 sec., normal 12 sec.; June 13, 1947, quick 20 sec., normal 11 sec.; June 20, 1947, quick 18 sec. normal 12 sec. The liver was slightly larger than normal, about two inches below the costal margin. The stools became yellowish intermittently, but the icterus index went up to 104 on May 12 and came down to 73 on May 16, and later remained around 50 from June 13 to June 20 (see graph). Urobilinogen was absent from the feces and persisted at 1:10 in the urine. The bile continued to be strongly positive in the urine. Urea nitrogen, total protein and cholesterol were all within normal limits as follows:

Blood Chemistry. May 16: Urea nitrogen was 14.4 mg.; on May 27, 18.1 mg. May 19: Total protein 6.6 gm., albumin 4.2 gm., globulin 2.4 gm. May 2: Total protein 6.2 gm. May 20: Total protein 6.1 gm. May 20: Cholesterol 222 mg. July 14: Cholesterol 250 mg.

Our impression at this time was that we were dealing with a congenital obstruction of the bile ducts. For this reason a surgical consultation was held.

It was the opinion of the surgical staff that we had a congenital atresia of the bile ducts, but in view of the pulmonary atelectasis and the age of the infant (two months old), they did not recom-

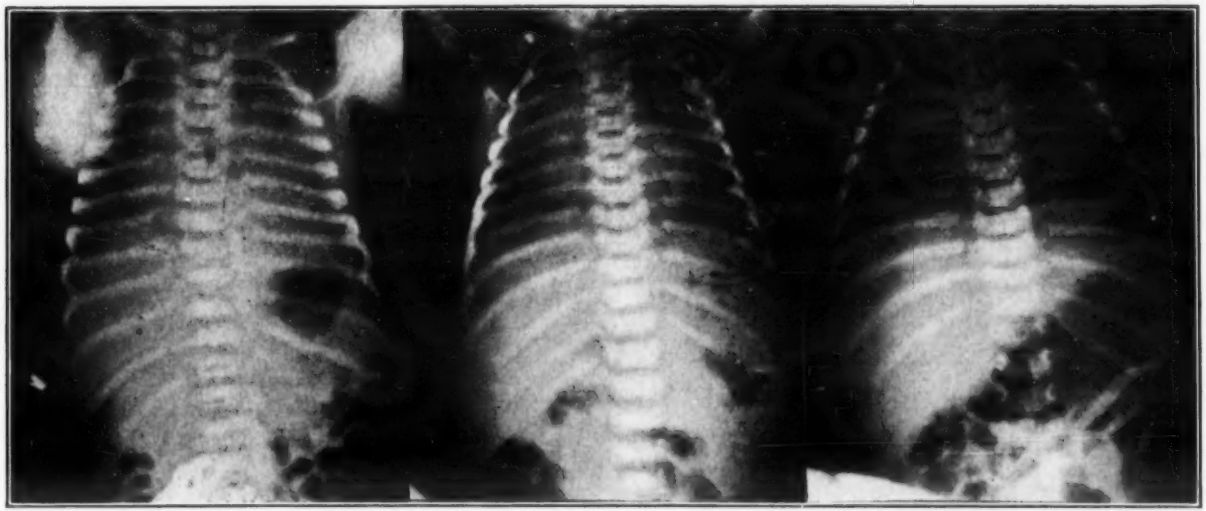

Fig. 1. Portable radiograph examination of the chest (May 7, 1947) shows an incomplete expansion of either lung field. Air filled bronchi are noted through the cardiac shadow. Findings—probable bilateral congenital atelectasis.

Fig. 2. Further examination (May 21, 1947) shows an area of absent aeration in the region of the right upper lobe. The balance of the lung fields is well aerated at this time. The heart is of fairly normal contour.

Fig. 3. Further examination (June 11, 1947) shows an opacity involving the medial half of the right lung. The left lung appears normally aerated. Findings are indicative of an atelectasis.

mend operation at this time. It was agreed that the infant should be older by several weeks before any surgical intervention.

The infant progressed favorably. There was an increase in weight. The skin was still dusky and icteric in color, but of good turgor. Stools were clay-colored. The urine was bile tinged.

Blood count on June 20, 1947: Hemoglobin 85 per cent, red blood cells 4,500,000, white blood cells 8,500, no erythroblasts.

The infant continued its stay in the hospital. At the end of

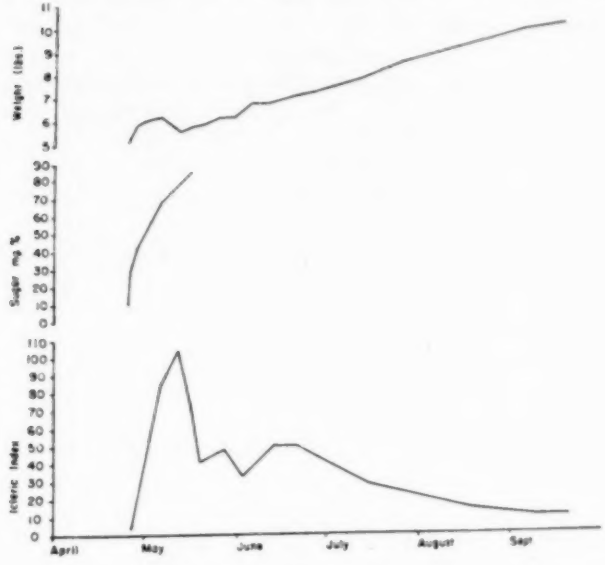

Fig. 4. Infant G. Weight, sugar and icteric index curves during entire hospitalization.

a two and one-half month's stay, the lung fields showed more expansion, the breath sounds came through fairly well even in the upper right lung bed.

The heart was regular, there was no murmur. The liver was not enlarged, the skin was still a yellowish tinge although of a much lighter color. Icterus index with wide variation stabilized around fifty from May 27 to June 26 (see graph).

Under continued treatment of bile salts by mouth the stools

became pasty and yellowish in color, and of good consistency. Urobilinogen in the urine was always below normal.

It was recommended at this time to discharge the infant to home surroundings bearing in mind that an exploratory operation might have to be done at some future date. However, in the following weeks, the infant rapidly gained in weight, the skin became clearer, there was a complete disappearance of the dusky color, and the lung fields fully expanded.

Icterus Index. The icterus index dropped to 28.8 on July 14, fourteen point five (14.5) on August 18, and finally, on September 18, to 10.5, which we considered a high normal.

Hemoglobin and red blood cells remained at about the same levels—hemoglobin in the upper eighty per cent and the red blood cells over 4,500,000.

At this point, it may be in order to consider the various signs and symptoms as they developed in this newborn. The first to be considered was the hypoglycemia.

HYPOGLYCEMIA

In infants of diabetic mothers, a period of hypoglycemia occurs 2 to 24 hours after birth. Instances of hypoglycemia in infants of non-diabetic mothers have been reported.[1] Most cases are probably due to an increased physiologic hyperactivity of the islets as a result of high sugar level in the mother.

Physiologic hyperactivity, or the Staub-Traugott phenomenon, where frequent ingestion of carbohydrate may result in a decided hypoglycemia, is not often seen at this age.

Of course, the most common cause of low blood sugar is insulin administration. Islet tumors are rare in infancy. However, in relative hyperinsulinism[2] failure of glycogen storage, particularly in the liver, or insufficient absorption of carbohydrate from the intestinal tract will precipitate hypoglycemia. Disease of the small bowel, such as diarrhea, vomiting, fistula, pyloric stenosis, with or without superimposed infection, produces a combination of decreased intake and increased utilization.

The central position of the liver in maintaining a constant blood sugar level should be stressed. Disease of this organ may bring about hypoglycemia by damaging the glycogenolytic or glycogenic function or by damaging storage capacity. Although only 20 per

cent of normal liver tissue is necessary to maintain adequate resting carbohydrate metabolism, sufficient liver destruction is seen in salicylate poisoning, acute catarrhal jaundice and severe infections so that hypoglycemia results. In Von Gierke's disease, there is abundant glycogen stored in the liver but it fails to serve as a source of glucose.

Lack of development of the regulatory mechanism in normal newborn infants results in a mild hypoglycemia during the first four or five days of life, and in them seems to be due to a state of relative hyperinsulinism. This probably was the underlying factor in our case. As his neuro-endocrine balance is not stabilized, the infant is at a greater disadvantage than the adult when carbohydrate supplies to the liver are reduced.

Endocrine imbalance[3] is a very important factor. Hemorrhage into the adrenals is not rare in newborns. This produces decreased epinephrin in the blood and deficient liver glycogenolysis ensues. Some children tend to have recurrent attacks of very severe hypoglycemia which probably are due to the lack of insulin antagonists. Failure of any one of the endocrines regulating the blood sugar level does not lead to fatal attack as there is reduplication of action which prevents grave hypoglycemia in hypothyroidism or destruction of the posterior pituitary. When, however, the cortex of both adrenals is destroyed, as sometimes happens in Addison's disease, there is a more severe hypoglycemia because not only is a diabetogenic hormone lost but a hormone accelerating the formation of carbohydrate from protein. The entire endocrine defense rests on the anterior pituitary. Therefore, in Simmonds' disease, there is a grave hypoglycemia as the thyroid and adrenal cortex suffer secondary degeneration.

Severe hemorrhage into the base of the brain is said to produce hypoglycemia.

A number of cases of hypoglycemia are due to an autonomic imbalance or "functional hyperinsulinism." These cases are characterized by a normal fasting blood sugar with a tendency for the level of the blood sugar to fall abnormally low from three to five hours after the ingestion of carbohydrates.

Holt[4] states, that cryptogenic hypoglycemia is the most important and most troublesome type. Holt noted, from a study of eleven cases, that the rare occurrence of hypoglycemic children with

recurrent convulsive manifestations is usually found between the ages of one and three years and is due to a functional hepatic disorder of an intermittent nature. The exciting cause is a temporary depletion of glycogen reserve usually due to insufficient food intake. It is also possible that the pituitary or adrenal cortex are at fault.

The most prominent symptoms of hypoglycemia are those involving the central nervous system. They appear first, remain throughout the episode and are the most dangerous. Studies indicate that profound hypoglycemia may depress cerebral metabolism to one-fourth the normal rate.

JAUNDICE

Before discussing the second cardinal sign, jaundice, which manifested itself on the fourth day of life, it may be of academic interest to review the embryology, pathology and physiology of the extra hepatic biliary system.

When the embryo is but four weeks old (2.5 mm.), an evagination called the hepatic diverticulum arises from the duodenal portion of the foregut. Its cephalic portion is solid and is destined to form the liver, while the caudal portion is hollow, continuous with the lumen of the gut, and is destined to become the extrahepatic biliary system. Constriction of the entire diverticulum occurs, so that the pars hepatica, while generating into liver lobules, is connected with the intestine through but a narrow cord of cells. It is generally believed that epithelial proliferation accounts for the loss of patency in these ducts, and that later these solid cords recanalize.

The liver parenchyma develops rapidly and the whole organ is comparatively huge at nine weeks, lobes being recognizable early. With the completion of the recanalization process of the pars cystica, the formation of the intra- and extrahepatic systems is finished and secretion and passage of bile occurs during the twelfth embryonic week.[3]

ETIOLOGY

The exact etiology of agenesis and atresia of the bile ducts is unknown. Simple failure of the hepatic diverticulum to form, and if it does form, simple failure of recanalization of the pars cystica,

is, of course, the most attractive theory of the etiology of agenesis and of atresia respectively.

Nutting and Wells[6], in 1945, reviewed the literature on the etiology of duct anomalies, and in addition to the attractive theory of Moolten presented the following for consideration: 1. Catarrhal inflammatory processes in the liver, descending into the bile ducts and causing their complete occlusion. 2. Absorption of toxins from the mother which injure the liver and bile ducts of the fetus. 3. Syphilis. 4. Mechanical traction of the mesentery, twisting the

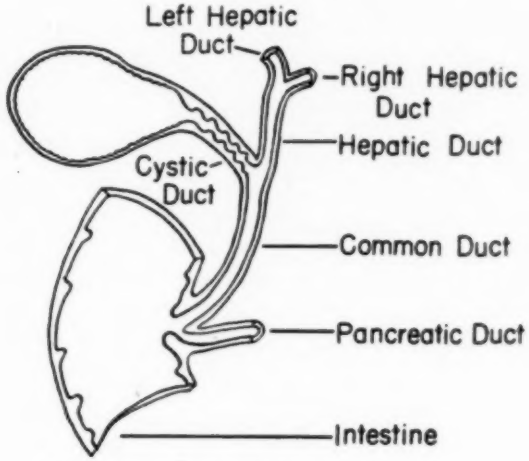

Fig. 5. Schematic drawing of normal bile system.

ducts. 5. Fetal peritonitis with adhesions and constriction of the ducts. 6. Abnormalities in vascular supply resulting in deformity of the duct.

PATHOLOGY

The malformations usually fall into one of Ladd's[7] classifications:

Group I. Cases in which there are no extrahepatic ducts.

Group II. Cases in which there is an atresia of the hepatic ducts.

Group III. Cases in which there is an atresia of the common bile duct.

Group IV. Cases in which the gall bladder is represented by a

moderately sized cyst not connected to the common bile duct and in which there may or may not be any common or hepatic ducts.

Group V. Cases in which the gall bladder connects directly with the duodenum but in which there are no other hepatic ducts. That is, no ducts connect the liver and gall bladder or the liver and intestine.

Group VI. Cases in which there is a stenosis of the common duct plugged with inspissated bile causing complete obstruction.

Group VII. Cases in which there is a narrowing of the common bile duct causing portal obstruction.

Closely associated with and secondary to obstruction of the duct system is biliary cirrhosis of the liver. So commonly is it seen that Nutting and Wells[6] advise liver biopsy in cases of undiagnosed neonatal jaundice to establish the diagnosis. Grossly and histologically, the liver is likened to that of pure portal cirrhosis, except that the bile canaliculi and bilary passage are loaded with bile pigment. The fibrous tissue radiates from the bile ducts, and not only surrounds but invades the liver lobule.

There are two main effects produced by complete extrahepatic blockage of bile, from which spring a multitude of interesting phenomena. These two effects are: 1. obstructive jaundice, with subsequent biliary cirrhosis, and 2. lack of normal digestion and absorption of fat and fat-soluble vitamins in the intestinal tract.

The characteristics of obstructive jaundice are well-known. Jaundice itself is due principally to suppression of secretion of bile in the liver, and not due to the regurgitation into the blood stream of bile already secreted. Secretion continues only until the pressure in the ducts equalizes the secretory pressure of the liver cells. Once that has occurred, bile passes from the blood into the liver cell, but cannot be secreted, and hence is absorbed into the blood stream and lymphatics.[8]

In obstructive jaundice, the hyperbilirubinemia tends to be pronounced with deep staining of the sclera and skin. The icterus index usually ranges between 50 and 200 units and the qualitative van den Bergh reaction is direct and immediate. There is an absence of bile in the stool most of the time, except in the rare instance when bile may be carried by the blood to the intestinal mucosa, and may appear in the intestines.[6] There is a highly positive test for bile in the urine.

In the liver, the process of cirrhosis may be progressive as a result of blocking of the portal venous flow, ascites may develop in the later stages of the course. Low total protein, and especially low serum albumin, contribute to the production of ascites and subcutaneous edema.

Bile is essential for the efficient digestion and absorption of fat by pancreatic and intestinal juices. While cleavage of the fat molecule may be accomplished with the aid of gastric lipase in the absence of bile, the transportation of the split molecule across the intestinal mucosa is greatly interfered with. Thus 75-85 per cent of ingested fat is found in the stools of these patients.

The most marked effect on vitamin absorption is seen in regard to vitamin K. Bleeding tendencies are frequently encountered. They are usually characterized by a prolonged prothrombin time, normal coagulation and bleeding time, and a normal thrombocyte count.[10] Poor liver function and mild splenomegaly may contribute to the production of this phenomenon.

Moderate anemia, most often microcytic hypochromic, is usually seen in these infants, and is the result of a combination of poor absorption from the intestinal tract, diffuse liver cell injury, mild splenomegaly and bleeding tendencies.

SYMPTOMS

Jaundice may be evident at the time of delivery, but usually manifests itself between the fourth and tenth days of life as in our case. The jaundice may become intensified for two to three weeks, after which time it remains the same depth. The range of icterus indexes in our case was between 18.2 to 100.2 units.

COMMENT

Erythroblasts[11] in blood smears at birth are not unusual in many cases of atelectasis or accompanying hypoglycemia.

Functional hepatic disorder may have been the primary basis for the hypoglycemia. As the infant's neuro-endocrine balance is not stabilized, the infant is at a great disadvantage when carbohydrate substances to the liver are reduced. The lack of development of the regulatory mechanism during the first five days of life accounted for the respiratory disturbance, atelectasis, as well as for the state of relative hyperinsulinism.

Since this was a newborn infant, and the mother was non-diabetic, we are certain that there was no excessive carbohydrate intake with sudden removal prior to the attack of hypoglycemia. We therefore feel that liver damage was the basis of this initial onset of hypoglycemia and the cause of the gradual onset of jaundice which became the outstanding clinical feature as the case progressed.

The cyanosis disappeared with the use of intravenous 10 per cent glucose in saline plus continuous oxygen. There was also a progressive expansion of the lung fields. The blood sugar levels from 10 mg. % increased to 62 mg. % at the end of one week, and succeeding weeks showed a steady rise.

Regarding the jaundice, at first we were almost convinced that we were dealing with a congenital anomaly. The icterus index was high. The stools were chalky, the urine amber colored. On several occasions, the icterus index fluctuated. This led us to believe that perhaps we might be dealing with a partial obstruction.

Surgery was considered, but, due to the age of the infant, the surgeons decided to wait. Bile salts were added to the infant's diet. At the end of the sixth week the icterus index began to drop (refer to chart). The infant steadily gained in weight.

We were fearful of the outcome in the event the case was one of congenital anomaly of the bile ducts. From experience we know that the prognosis of these cases is not good. If so, the average duration of life is approximately five months. Ladd[7] reporting his series of cases, showed 15 per cent survival.

As the weeks went by, the skin became lighter, the icterus index dropped gradually and is now 10.2, considered by some a high normal. The infant has yellow pasty stools. Weight on January 3, 1948 is fifteen pounds ten ounces.

SUMMARY

An instance of obstruction of the bile ducts, hypoglycemia and atelectasis with a comprehensive review of the literature with relation to the symptoms is included.

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LAURENCE-MOON-BARDET-BIEDL SYNDROME. (Revista Clinica de São Paulo, 23: 1, Jan.-Feb. 1948). The authors report a typical case of Laurence-Biedl syndrome in a girl 17 years old. Consanguinity of the parents and ancestors and heredity were ruled out. Administration of thyroid in daily doses of 0.1 mg. for ten consecutive days followed by ten days without therapy for several months normalized the metabolism but had no influence on the syndrome. Roentgen examination of the head showed a normal sella turcica of increased size. Administration of thyroid for some time resulted in an abundant elimination of gonadotropin in the urine. These facts show that the syndrome is not of hypophysial origin. The authors believe that the syndrome and the congenital malformations associated with it are due to defects of the germ plasm.—*Journal A.M.A.*

FORECASTING SMALLPOX EPIDEMICS ON CLIMATIC DATA. (Journal of Hygiene, London, 46: 19, March 1948). Rogers shows that earlier work on forecasting epidemics of smallpox in India, based on the study of the previous rainfall and humidity, has been confirmed and extended. An extension of the inquiry to other tropical countries indicates that in tropical countries in general with well marked dry and wet seasons, the annual decline of smallpox incidence to a low minimum in relation to high absolute humidities during the rainy season is the rule; for it is absent in equatorial Uganda with rains throughout the year, and both the monsoon period and the monthly incidence of smallpox are reversed in such a country of the Southern hemisphere as Nyassaland in accordance with the general rule. Where smallpox is endemic, increased prevalence in any year may usually be foreseen by watching the meteorologic records in time to control the expected increase to some extent by increased use of vaccination.—*Journal A.M.A.*

MULTIPLE NEURITIS AND POLIOMYELITIS

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The failure of the virus theory of poliomyelitis adequately to explain the epidemiology of the disease requires reappraisal of facts that had been presented preceding the discovery of the "virus." The writer[1] has already pointed out in a previous report that the paralysis produced by the injection of nervous tissues from poliomyelitis victims into experimental animals—originally observed by Landsteiner,[2] and which forms the basis of the virus theory—can also be produced by the injection of normal homologous and heterologous nervous tissues. This fact alone would strongly lead one to suspect that unjustified conclusions have been formed that have resulted in our present state of confusion regarding acute epidemic poliomyelitis.

Prior to 1908, the two principal theories regarding the causation of poliomyelitis were: 1, that it is caused by an infectious agent; 2, that it results from an exogenous poison. The infectious agent theory has been exhaustively investigated with extremely discouraging results. But the poison theory of the cause of poliomyelitis has never adequately been investigated. Since Landsteiner's discovery of a "virus" (1908) in connection with this disease, the poison theory of cause has been almost ignored.

The confusion that exists in regard to the epidemiology of poliomyelitis and the present reliance on theoretical considerations, the inability to prevent this disease or adequately to treat its victims, result from a number of reasons. It is the purpose of this report to indicate some of these reasons and to show emphatically that poliomyelitis not only is not a modern disease but that diseases apparently unrelated topographically are intimately related.

MULTIPLE NEURITIS

The recognition of multiple or peripheral neuritis as a clinical and pathological entity dates from the publications of S. Weir Mitchell,[3] Dumenil,[4] and van Leyden.[5] Cobb and Coggeshall[6] define neuritis as a painful degenerative process of the peripheral neurone causing a functional loss which, according to the anatomy involved, may be sensory, motor or mixed. They point out that

rarely if ever are pathological changes found in one part of the neurone alone; if the axone is primarily affected there is soon a change in the nerve cell and vice versa. When the primary pathological condition is localized at one part of the course of the nerve fiber, its position is often indicated by the symptomatology; eventually, however, the whole neurone suffers. The element of pain may be extremely mild and evanescent as in lead poisoning and infantile paralysis. Frequently it has been pointed out that the term "multiple neuritis" is a misnomer, since in addition to the degeneration of the peripheral nerves, there are often changes in the cells of the anterior horns and the muscles. The term "neuronitis," or, if multiple lesions are present, "polyneuronitis" has been suggested to replace the terms neuritis and multiple neuritis.

It is generally agreed that multiple neuritis is the result of exogenous and endogenous poisons. It will be pointed out by the

TABLE 1. *Chemical Substances Known to Produce Multiple Neuritis in Man and Experimental Animals.*

Alcohol Ethyl	Carbon tetrachloride	Gold	Selenium
Alcohol Methyl			
Amidopyrine	Chlorbutanol	Lead	Silver
Amylene hydrate	Chloral hydrate	Mercury	Sulfonal
Aniline	Chloretone	Methyl chloride	Sulfonamides
Antivitamins	Coal tar products	Morphine	Sulfonethylmethane
Apiol	Copper	Nitrobenzene	Thallium
Arsenic	Cyanide	Oxalates	Trichlorethylene
Barbiturates	Ether	Paraldehyde	Trinitrotoluene
Bismuth	Fluorine	Phenacetin	Trional
Carbon bisulphide		Phenazone (Antipyrine)	Triorthocresyl phosphate (Jamaica ginger)
Carbon monoxide			

writer of this report that many of the cases of multiple neuritis that are attributed to endogenous poisons probably originate from exogenous poisons, the manifestations of which are merely precipitated by other factors, such as infections, pregnancy, operations, etc.

Some of the chemicals that are known to produce multiple neuritis when they are introduced into the body of human or experimental animals are listed in Table 1.

Many of these chemical substances can cause death within a few hours or days if taken in adequate amounts, whereas, other chem-

icals, such as carbon monoxide and cyanide, can result in some cases almost instantaneously in death. Yet, many of these chemical substances, including the deadly poison cyanide, have therapeutic merit and in small quantities have beneficial effects. Cyanide was employed in the past in the treatment of coughs and at the present time is found in syrup of wild cherry (Prunus virginiana). "It is also present in the tincture of chloroform and morphine, B.P."[7]

Despite the fact that a great diversity of poisons is capable of producing multiple neuritis, nevertheless the contributary factors, symptoms, pathological lesions, etc. are essentially identical.

The young are particularly susceptible to poisons; young adults are more susceptible than their elders. Babies and children react to smaller doses than adults. Some authors report that attacks of lead poisoning are more common in the summer than in the winter. Individual susceptibility to the effect of poisons varies greatly. Thus, as Wechsler[8] points out: "Untold numbers of persons partake of alcohol to excess and only a few develop polyneuritis. An infinitesimal number who receive arsenic develop peripheral neuritis." Similar observations have been made in regard to lead and other poisons. It is axiomatic that not every person exposed to the same chemical agent will respond in the same manner.

"There is some evidence to show that certain families are more susceptible to the influence of lead than others."[9] A significant remark made by Walshe[10] concerning polyneuritis corresponds with observations made during epidemics of poliomyelitis: "The illness usually develops in perfectly healthy individuals* suffering from no discoverable infection or dietary insufficiency. . . . To ascribe the illness to B-avitaminosis has as yet no justification."

In lead poisoning, "any sudden unusual exposure, a drinking bout or an infection, such as influenza, may produce acute symptoms."[11] Such factors also appear to be important in other forms of poisoning. It has been pointed out that acidosis may precipitate an acute attack of multiple neuritis. Cobb and Coggeshall[6] feel that subacute abscess formation may be a factor. Fatigue, general or

*Although poliomyelitis is frequently stated to occur most frequently in healthy individuals, such an individual may be healthy or merely appear to be healthy. Individuals with varying degrees of hypothyroidism would tend to be robust and often healthy appearing, whereas, the hyperthyroid type is ordinarily the opposite. Carlson[12] has well stated: "Who is healthy and when?" Neither can we be unmindful of the dictum of Professor Halbane[13] in his study of biological phenomenon: "We must keep the whole organism in view."

localized, has frequently been mentioned as an important factor in the various forms of multiple neuritis.

The form in which the poison enters the body is an important factor in any case. The mode of entrance of the poisons into the body is by inhalation of vapors, fumes or dusts, absorption through the skin, and ingestion by mouth. In experimental animals, injection of some of these poisons will produce effects, and often more rapidly, than by other routes.

Stewart[12] points out that in multiple neuritis there is: "1. *The spontaneous onset of symptoms*, which may develop rapidly or slowly, 2. *The evolution of the clinical picture*, which is characteristic and occurs in three stages, (a) *a stage of invasion*, (b) *a stage of elaboration*, in which the symptoms and signs are fully developed; this may end in the death of the patient, or, more frequently, (c) *a stage of recuperation* which may be prolonged, and 3. The symmetry and bilaterality of the symptoms in each individual case as regards, (a) their distribution, (b) the character of the functional disturbance, (c) the time and order of their recovery."

In cases of lead poisoning the symptoms are more or less representative of those observed in other forms of multiple neuritis, including epidemic poliomyelitis, and, therefore, these will be considered.

In acute forms of lead poisoning the symptoms are those of acute gastro-enteritis which include epigastric pain, nausea, vomiting and diarrhea. In subacute and chronic forms of lead poisoning there is a tendency to indigestion, loss of appetite, nausea, abdominal uneasiness and constipation. There may be lethargy and weakness. There may be headache and vague pains in the back and limbs, most particularly in the region about the joints. There is often abdominal colic, diffuse in distribution, but most marked in the middle of the abdomen. There may be vomiting and there is usually constipation. The temperature is slightly elevated and the blood pressure is increased.

Although the paralysis which occurs in lead poisoning is described as bilateral, cases of unilateral wrist-drop have been observed. This peculiar distribution of paralysis in lead poisoning has been ascribed to the flexion and extension of the wrist by painters; it has been pointed out that it is more common in the right wrist than in the left. The element of fatigue of the wrist

muscles is considered an important factor in the localization of the paralysis. The legs are not uncommonly affected in children. "Any motor nerve, including the cranial motor nerves, may show involvement. Some degree of atrophy usually develops and the muscles show at least partial reaction of degeneration."[9] Cases of lead poisoning with identical findings as those found in acute anterior poliomyelitis have been described by Adamkiewicz,[15] Putnam[16] and others. The paralysis in all forms of multiple neuritis is characteritically flaccid. Ford[17] points out: "No doubt, various individuals differ in their reaction, for exposure to the same poison will produce encephalopathy in one patient and merely polyneuritis in another; while a third may combine both symptom complexes or even develop the picture of Landry's paralysis."

A leukocytosis of 10,000 to 15,000 cells is often present, and higher values are not unknown in multiple neuritis. Although spinal fluid changes consisting only of increased pressure and an increase in globulin have been described in cases of multiple neuritis, including epidemic poliomyelitis, yet, both the latter and lead poisoning may show a pleocytosis. In the case of lead poisoning, these spinal fluid changes have been found consistently by a number of workers in encephalopathic patients. Aub et al.[11] state regarding this fact: "The question, therefore, arises whether lead encephalomyelitis is not really a meningo-encephalopathy which is primarily a chronic productive meningitis, characterized during acute exacerbations by a degree of round cell infiltration varying with the severity of the attack." Mosny[18] divides lead meningoencephalopathy into four classes—acute, subacute, mild and latent—and believes that he can distinguish between them by the spinal cell count. If cases of multiple neuritis, other than epidemic poliomyelitis and lead poisoning, were studied as intensively in all stages of development, similar spinal fluid changes would doubtless be found.

In the neuritis of herpes zoster (acute posterior poliomyelitis)*, for example, there is frequently present a pleocytosis.

*An increased incidence of herpes zoster (acute posterior poliomyelitis) has been observed during epidemics of acute anterior poliomyelitis. Many of the features, including occasional flaccid motor paralysis, are similar to those of the latter disease. Although herpes zoster is usually attributed to a virus infection, yet cases have commonly resulted from poisoning with some of the chemical substances mentioned in Table 1. The so-called "virus" of this disease would appear, therefore, to be formed or activated in the human body in such cases as a result of the poisoning.

The pathological changes in all cases of multiple neuritis, regardless of the cause, are found in the spinal cord and these consist in degeneration of the anterior horn cells with vacuolization of the ganglion cells, perinuclear chromatolysis, fatty degeneration and marked shrinking of the cells.

Wechsler[19] points out that every case of multiple neuritis, regardless of the cause, can be treated more or less uniformly. The patient should be put to bed, he advises, at least until it is definitely established that the condition is not progressive and there is no danger in being up and about. "Hot air and hot packs may add greatly to the comfort of the patient, improve the circulation, and promote recovery. . . . The question of contractures must not be overlooked. They generally make their appearance late, but attempts should be made to avoid them from the beginning."

The therapeutic agents that have been employed in the treatment of multiple neuritis include iodides, calcium, ascorbic acid, methylene blue and sodium thiosulphate.

BERIBERI

The preceding facts concerning multiple neuritis in general apply specifically to beriberi. This disease has existed for centuries. In the past it has been attributed to a number of factors, including moist weather; unfit water and soil; night air; poisoning by arsenic, carbon dioxide, oxalates, rice or fish, autointoxication; nitrogen starvation; phosphorus starvation; bacterial toxins; vitamin B_1 deficiency, etc.

In 1896, Eykman[20] concluded from his studies on beriberi that a poison* exists in rice and for this poison something in the pericarp is an antidote. He considered this poisonous substance to be the cause of beriberi but he was unable to identify it. Many investigators have since strongly emphasized that a toxic substance is responsible for beriberi. It has been pointed out that when rice is cured, i.e., parboiled before it is decorticated, beriberi is pre-

*Many grasses, including the cereal grasses, rice, corn, wheat, etc., as well as many fruits and vegetables, contain a cyanophore glucoside capable of producing hydrocyanic acid on hydrolysis. The pericarp of the rice contains not only vitamin B_1 but also minerals, including iodine. Beriberi has usually been identified with rice eating peoples but it has been observed in countries producing corn and other cereals. Messerle[21] produced experimental beriberi in birds with hydrocyanic acid.

Oxalic acid and other dibasic acids are widespread in the plant kingdom in the free state and in the form of salts. Oxalic acid can be obtained by spontaneous hydrolysis of an aqueous solution of cyanogens.

vented. However, when uncured rice is eaten after it is decorticated, beriberi may develop.

In 1911, Funk[21] described a crystalline base obtained from an extract of rice polishings which was effective in curing pigeons suffering from multiple neuritis that had been induced by a diet of polished rice. He designated this substance as a *vitamine* (*vita* meaning *life*, and *amine* indicating *nitrogenous*). In subsequent reports he found purine and pyrimidine derivatives also to be effective in the treatment of beriberi. Jansen and Donath[22] isolated crystals from rice polishings and Williams[23] suggested that this vitamin B_1 be called thiamin, a term now generally employed. This work constitutes the basis of the "vitamin" theory of beriberi. However, it has been shown, as will be pointed out later, that beriberi can be cured without employing vitamin B_1 in its treatment.

A perusal of the medical literature shows that there exists in the minds of a number of writers the possibility that beriberi and poliomyelitis are identical diseases. The present writer agrees with these views of identity but is convinced that both originate from the same exogenous poison in food and water.

Chantemesse and Ramond (1898)[25] and Huffman (1911)[26] called attention to the resemblance of epidemic poliomyelitis and beriberi. Braddon (1907)[27], in his book on beriberi, states that these two diseases are related genetically. He mentions several other investigators of epidemics of beriberi who had made similar observations. Helms (1941)[28] pointed out the similarity of these diseases and McCormick (1938, 1942, 1943, 1944),[29-33] in several reports, showed that there is a very definite relationship between epidemic poliomyelitis and beriberi. He suggested that the so-called "virus" of the former is an endogenous by-product of the disease.

Van Rooyen and Morgan[34] were perhaps the first to designate epidemic poliomyelitis as a *military disease* in the Middle East. McAlpine[35] points out that British and American troops serving in India and the Middle East in the recent World War showed a higher incidence of poliomyelitis than the armies at home. He says: "They do not appear to have shared the high immunity to the disease shown by the native populations, presumably because the strains of virus to which the troops were exposed differed from those in their home countries." In regard to the so-called "virus" of

this disease, Paul, Havens and Van Rooyen,[36] found in a series of 35 cases of epidemic poliomyelitis in the Middle East among military personnel, that the "virus" was present in the stools of 9 out of 10 *fatal cases*. Negative results were obtained in 5 non-fatal cases and in 20 atypical cases. The striking fact in all of the reports is that inquiries revealed that epidemic poliomyelitis was said to be rare in the native population. These facts suggest that an identical disease entity was encountered which was designated as beriberi in the native population and as acute anterior poliomyelitis when it occurred in the troops in the same region. The modern concept of a vitamin deficiency disease as the cause of the former and a "virus" infection for the latter disease, therefore, precludes the identity of a single disease entity. The supposed limitation of beriberi to the Orient and of poliomyelitis to temperate zones naturally adds to the confusion. Although the feeding habits of the two groups, troops and native population, might have been different, yet, the fact remains that the source of the water supply would necessarily be identical.

This common factor is best illustrated by considering the conditions under which rice is grown and subsequent factors in relation to the growing of rice. This subject will be considered again in relation to "miasmic poisoning."

"Rice, the only cereal food available to tropical and Oriental countries, is a *swamp dwelling grass* originally native to south-eastern Asia, cultivated in China and India for at least 4000 years. From there it spread to Syria and North Africa, reaching Europe in the fifteenth century and America in the seventeenth when it was brought to North Carolina."[37] Rice is easily grown in low river flats which are subject to frequent inundations that leave behind them a deposit of rich alluvial soil. The main crops of rice throughout the world, therefore, are found to be associated with streams that flood their banks. In the cultivation of rice, it is not only necessary to "capture" the water for the growth of the rice but also to release the water into the stream during the planting and harvesting seasons. Thus, stagnant water concentrated by evaporation and highly potent with decaying vegetation re-enters the streams which are likewise often employed for drinking purposes.

The raising of rice is not confined to the Orient. "In the United States, rice is grown in Louisiana, Texas, Arkansas and California. —the Sacramento Valley of the latter being particularly well fitted for its cultivation."[37] The 1946 World Almanac lists in addition to the above states, South Carolina, Mississippi, Georgia and Florida as rice producing states.

The first reference to poliomyelitis that appears in the United States is that reported by Colmer[38] in the parish of West Feliciana, Louisiana in the fall of 1841. His report suggests that the disease was endemic in this region. Multiple neuritis, which until recent years has been considered as merely an affliction from inflamation of the peripheral nerves rather than the more inclusive term "polyneuronitis," was first described in epidemic form in the United States by Bondurant.[39, 40]

In 1896, he described an epidemic in the Bryce Insane Hospital at Tuscaloosa, Alabama. He says: "During the ten years ending February 1895, there occurred not more than a dozen cases of multiple neuritis, and all of these with two exceptions were of the alcoholic or syphilitic variety. Between February 1895 and February 1896, thirteen other cases happened in a hospital population of about 1,200 for none of which a definite cause could be found. After a period of immunity, the disease reappeared in the late summer of 1896, when, following a season of unusual dryness, 58 patients were attacked, 53 of these occuring in September and 5 in October. There were in all 71 cases in a population of 1,200. Bondurant, after this outbreak, concluded the disease to be identical with beriberi and so described it. In the second epidemic there were more acute cases; nearly all had fever; some had considerable pyrexia.

Bondurant ascribed the cause of these outbreaks, with very convincing reasons, to something in the *water supply*. He points out that the institution had used its own spring for 34 years but at the time of the outbreaks recourse had been made to the use of river water in addition to the source from the spring. Water had been pumped up directly into the hospital tank. When this source of water was employed, there was an outbreak of the disease coinciding with its use both in time and very closely in extent with the amount of water taken from the stream.

Bondurant quotes a description of an epidemic supplied to him

by Dr. W. B. Barner, Assistant Physician of the Arkansas State Insane Hospital, Little Rock. At this institution, a small epidemic of cases similar to the Tuscaloosa ones occurred in July, August and September of 1895. The cases numbered between 20 and 30. The gravity of the cases varied, the milder ones showing edema of the ankles with ill-defined paralytic symptoms, the severe ones complete motor paralytic symptoms. Only one case proved fatal. The temperature ranged between 100° and 102° F. in this series of cases. Bondurant claimed that these cases were beriberi.

Caverly[41] described an epidemic of "infantile paralysis" in Rutland County, Vermont, which occurred from June through August 1894. There were upwards of 130 cases with almost all of them occurring in one valley, at the end of a very dry season. The water supply was derived from a stream that traversed the valley, then very low, and sewage contaminated, as it was in other epidemics he details. In this epidemic most of the cases occurred in children; many domestic animals, including horses, dogs and fowls were also afflicted. Dr. Caverly wrote to Dr. Bondurant: "There is no doubt some of the cases in our epidemic were neuritis."

A similar outbreak, on a small scale, occurred, Bondurant states "during the summer of 1896, in Greene County, Alabama, about 40 miles south of Tuskaloosa." There were 15 or more cases scattered over an area of country 10 to 15 miles in diameter. Adults, as well as children, were attacked. The disease began with fever and systemic manifestations. A few days to a week after the fever appeared some paralysis followed.

Young (1903)[42] recorded cases of beriberi which occurred in Abbeville, Louisiana. During the five years preceding 1903, he states that he had met with many cases of beriberi in that region. He mentions three physicians who had recently treated thirty, six and six cases, respectively, and all agreed as to the diagnosis. The cases were all under observation within a period of 24 months, and within a radius of 20 miles. He points out that the cases occurred without exception in a locality surrounded by rice fields.

Pollock (1923),[43] in describing multiple neuritis, points out the occurrence of this disease during epidemics of poliomyelitis and states concerning it: "Mention should be made of acute pernicious multiple neuritis which comes on suddenly, progresses rapidly and causes death in a few days or weeks." These cases show the ordin-

ary symptoms of neuritis paralysis and probably are due to the same cause as poliomyelitis, occurring with this disease in epidemics. Braddon (1907)[27] emphasizes that acute anterior poliomyelitis and beriberi are allied genetically.

It would consume too much space in this report to compare the epidemiology, symptomatology and pathological affinities of beriberi and poliomyelitis. This comparison has been well made by several writers, especially by McCormick.[29-33] In fact, if one reads a description of one of these diseases in any standard textbook, he is reading essentially a description of the other. Braddon[27] points out that the course of beriberi is variable, even extremely so. Landon and Smith[44] make a similar remark concerning poliomyelitis. "It will be observed," they say, "in our description of the symptoms of the first stage that there is, so far, no symptom or group of symptoms which we may employ in stamping the condition as a clinical entity." Braddon[27] makes the following significant remark: "Whenever beriberi is most prevalent, the general sickness rate from other disorders also is usually much increased."

PELLAGRA

A long list of etiological factors have been given to account for epidemics of pellagra. Harris[45] lists no less than 48 theories, including the modern nicotinic acid deficiency theory which resulted from the work of Elvehjem et al.[46] in 1937. Among the theories that are mentioned is that of Jansen (1788)[47] and Titius (1791).[48] They suggested that "miasmic poisoning" is responsible for the disease. This theory has long since become obsolete. Yet, it appears to the writer that the miasmic poisoning theory is of utmost importance in understanding the relation of multiple neuritis and the related diseases.

Casal (1725),[49] a physician in Spain, was the first to describe cases of pellagra. He regarded it as *scorbutic leprosy* and considered the disease to be caused by a toxin in corn. Lombroso (1869),[50] the great Italian pellagrologist, conducted experiments in both man and animals and came to the conclusion that pellagra is caused by a toxin in fermented maize. The association of pellagra and corn, especially spoiled or fermented corn, has been emphasized by many workers since Casal's time. Clark[51, 52] and Barondes[53] have emphasized that the toxic substance in corn

responsible for pellagra is a cyanophore glucoside or nitrile which is capable of producing hydrocyanic acid on hydrolysis.

All of the toxic theories, regardless of the most convincing evidence, have been more or less ignored, as in the case of beriberi and poliomyelitis, only to be superceded by theories that do not adequately explain the disease. In regard to the corn toxin theory, Harris[54] remarks significantly: *"Men's opinions may change but facts remain the same always."*

Although pellagra usually has been and is considered to be endemic and more or less confined to the southern part of the United States, the evidence appears to indicate that it is widespread throughout the country. Strangely enough, the first case of pellagra was observed in the north.

This case was one reported by Gray[55] in Utica, N. Y., in 1864, and another case in the same year was reported by Tyler[56] in Somersville, N. Y. In interviews with two former guards of Confederate prison camps, Niles[57] was told that the men had a supposed eczema. Sherwell[58] of Brooklyn, in 1883, described a case of pellagra in an Italian sailor. In 1889, Bemis[59] left a written diagnosis of pellagra in the case of a white woman, a patient in the Charity Hospital, New Orleans. In 1902, Sherwell[60] reported another case of pellagra. In the same year, Harris[61] of Atlanta, Georgia, reported a case of pellagra with hookworm disease. In 1907, Searcy[62] reported an outbreak of pellagra in the Mt. Vernon Alabama Asylum for negroes. In the same year Babcock[63] reported pellagra in the inmates of the State Insane Asylum at Columbia, South Carolina. In 1907, Searcy[64] discovered its existence among the patients at the Bryce Hospital at Tuscaloosa, Alabama. In 1908, Lavinder[65] of the United States Public Health Service had his attention called to the disease by local physicians in the south. In 1906, the South Carolina Board of Health held a conference on pellagra. In 1909, the first Pellagra Conference was held in Columbia, South Carolina.

"At every contemporary medical meeting in the south, pellagra became the live topic for discussion, diagnoses on many cases previously regarded as psoriasis, eczema, syphilis, symmetrical gangrene, dermatitis, hookworm, dysentery, etc. were revised"[66] Dunn (1917)[67] states significantly concerning pellagra: "It is unfamiliar to many physicians and has little meaning to those in that community."

It appears to be noteworthy that in the same year, 1908, when epidemics of pellagra first began to be noticed in the southern states, and 13 other states also reported its existence, the poliomyelitis "virus" was discovered. Within a year the number of states reporting cases of pellagra had doubled. Pellagra has now been reported in every state in the Union.

The epidemiological features of beriberi, epidemic poliomyelitis and pellagra, except for the commonly considered regional distribution, are identical; all of these diseases occur in epidemics in the same season of the year; children and young adults are chiefly affected; rural cases exceed in number urban cases; these diseases have not proven to be infectious or communicable; doctors, nurses and other attendants, brought into intimate contact, do not contract these diseases; these diseases may be highly prevalent in one community and almost entirely absent in an adjoining community, and this for not one season alone, but for periods of years; each disease has been at one time or another considered to be infectious and not infrequently a "virus" has been mentioned as a causative factor; the incidence of all of these diseases is most prevalent along watercourses; investigation in each disease has resulted in essentially the same negative results.

Shelley[68] pointed out that cases of pellagra are most numerous near moving water. Sambon[69] was of the opinion that there was a definite relationship of pellagra to streams of water. The disease is most prevalent in the United States during years of drought following floods along waterways, especially along the Mississippi and its tributaries.

Goldberger and Sydenstricker (1927)[70] investigated pellagra in the Mississippi flood area. They state: "Notwithstanding the very general character of most of the information available, we are satisfied that in the localities visited in Tennessee, Arkansas and Mississippi, the incidence of pellagra is abnormally high. The overflow area of Tennessee, Arkansas and Mississippi is normally an area in which pellagra has been quite prevalent ever since the disease was recognized in 1908 or 1909."

Cases of beriberi and pellagra have been reported in the same individuals and in pellagra districts beriberi has been found to be associated with that disease. Combinations of these diseases are probably more common than reports in the literature would in-

dicate. Ringrose et al.,[71] as well as Mason,[72] have reported a pellagra-like syndrome in range chickens that were maintained on an adequate ration. The fact that they were given water ad lib is of interest. The work of Ringrose et al. was reported at a time (1930-1931) when an extensive epidemic of poliomyelitis swept the country and an extensive drought was prevalent.

Spies et al.[73] found that beriberi most commonly manifests itself as polyneuritis and frequently is associated with pellagra. Farnell and Yacovley[74] point out that pellagra is quite essentially a disease of the nervous system as is beriberi. They describe in detail the similarities of beriberi and pellagra and state: "We consider that the heretofore classical entities, pellagra and beriberi, are in general similar and that herein is the cause of many diagnostic errors. These errors have only metaphysical interest, for in pellagra there is always more or less beriberi and beriberi rarely exists without pellagra."

Mendelson[75] described a case of combined pellagra and bériberi and pointed out that in the Federated Malay States such cases are not uncommon. Castellani[76] notes that in some countries, where beriberi and pellagra are endemic, cases are met with in which, in addition to the beriberi symptoms, skin lesions are seen in the latter disease. He mentions the usual type of beriberi and a type with chronic roughening and pigmentation of the skin of the exposed parts. During the poliomyelitis epidemic of 1944 in New York City, Reyes[77] found skin lesions in 98 per cent of his cases His description of these skin lesions strongly suggests the presence of both poliomyelitis and pellagra.

The prophetic remarks made by two of the earlier writers, one concerning beriberi and the other referring to pellagra, are of importance in view of what has been pointed out and evidence which is to be presented. Manson (1901)[78] stated": . . . there is a considerable body of evidence tending to show that beriberi has spread into regions hitherto believed to be immune and even into Europe and other temperate climes. . . . It is possible, therefore, that in time an epidemic disease resembling beriberi, if it be not true beriberi, may attain permanent lodgement in temperate climates and thus add another burden to countries already sufficiently afflicted with their own indigenous diseases." Castellani[79] considered it probable that before many years, "pellagra will be found

to be prevalent all over the world." Lombroso,[50] who made the first intensive study of pellagra, said: "It is possible, of course, that more than one morbid entity may at present be included under the pellagrous syndrome."

MIASMIC POISONING

"Miasma" is a term that was formerly applied to malaria, which was supposed to originate from mists arising from swampy ground. However, the more inclusive terms, "miasmic poisoning," "malarial poisoning," "malarial fever" and "swamp fever" are found in the early literature and refer to diseases associated with swampy regions. Prior to the discovery of the malarial parasite in 1880 by Laveran,[80] a large group of diseases was described under the above terms. After the discovery of the malarial parasite many of these diseases appear to have been classified as "virus infections",* "vitamin deficiency diseases", etc.

In order to gain a clear conception of the apparent reasons for the confusion that exists today in determining the cause of some of our diseases, one can profitably retrace his steps to the era of confusion—the miasmic period—that existed prior to the discovery of the cause of malaria. The elaborate and exhaustive medical reports of the War of the Rebellion, which include observations in the field and in the hospitals during the years 1861-1865, and contemporary reports in medical journals, supply a rich source of information. In these reports we find that beriberi, pellagra and poliomyelitis doubtless existed, not only together and in various combinations in the same districts, but originated from the same cause, *swamp water*.

The fact that at least two main groups of diseases existed during the Civil War is indicated by Major Charles Smart[83] when he states: ". . . the clinical records of the war contain but few cases of malarial disease, but this absence of details is not surprising in view of the great prevalence of malarial fevers. . . . Although neuralgia was frequently regarded as a clinical associate of malarial fevers, or indeed as the legitimate offspring of the malarial poison, the cases reported on the monthly reports had no autumnal tides

*The application of the term "virus infection" to disease was not employed until after the work of Beijerinck (1898)[87] on mosaic disease in tobacco plants and Loeffler and Frosch[82] on foot-and-mouth disease in animals had been reported.

of prevalence to indicate their connection or dependence upon the cause of the paroxysmal fevers. To account for this we must assume that all neuralgic cases in any way connected with malaria must have been reported under the heading *miasmic diseases*, or which seems more probable, that neuralgia was less frequent in its association with malarial disease than was currently supposed." S. K. Towle,[84] Surgeon 30th Mass. Vols., in his notes of practice in the U.S. General Hospital at Baton Rouge, Louisiana during the year 1863, in alluding to the complicated character of the diseases observed in that hospital, states: "Indeed, the symptoms of many of the cases would indicate rather a combination of diseases than any one disease."

The frequent reference to epidemics of nausea and vomiting, at times associated with diarrhea are noted by Smart[83] and also by Mott.[85] Both acute and chronic forms of diarrhea are prominently mentioned, some of which were reported as dysentery. Mott states: "It was observed that records of 119 epidemics of dysentery showed that 62 began during or just after the occurrence of moist weather while 57 arose and ran their course during persistent dry weather." It is interesting to note in connection with the miasmic diseases that are being considered that Harris[86] observed that the dysentery which prevailed in Mifflin County, Pennsylvania, during the summer and fall of 1797, 1798 and 1799 spared the inhabitants of those districts in which the soil rested on limestone formation.

It is noted in the Civil War reports that many cases of palsy, chiefly paraplegia followed this so-called acute and chronic dysentery. Woodward[87] points out that 2,907 soldiers were reported to have been discharged from the service during the war on account of paralysis, and 272 deaths were attributed to the same cause, but no means existed of ascertaining how many cases were connected with dysentery.

Smart[83] states: "Among the cases of disease due apparently to exposure to cold and wet were many in which the spinal cord and its membranes became affected." Surgeon J. E. Sanborn,[88] 27th Iowa, writing from Jackson, Tennessee, March 31, 1863, also refers to this spinal affection as follows: "A singular affection has manifested itself in this regiment, taking a form of a severe spinal irritation and, possibly, spinal meningitis. There is pain in the

lumbar region, occasionally sharp but usually dull and aching; at times the feeling is described as a sense of weakness, with inability to stand or sit straight and a difficulty in lying down except in certain positions. There is tenderness on pressure upon the spinal processes or just upon either side of them. The first cases of this disorder I suspected to be feigned, but the prevalence of the affection and the character of many of the subjects soon indicated that in many cases it was a reality. Some cases were at first thought to be the result of an affection of the kidneys brought on by lying on the wet ground, an idea suggested by the fact that they were accompanied by *dark or very red urine*. In other cases it was observed that this affection either followed or accompanied chronic diarrhea. A number, having limped about with canes to support their bending spines for sometime, have finally been discharged."

Smart[53] says: "Sometimes the attack was so sudden that the case was reported as one of paralysis, or as inflammation of the spinal cord or its membranes, if associated at its inception with febrile movement and tenderness over some part of the spine. The disease was reported as chronic myelitis when the spinal tenderness was not associated with a symptomatic pyrexia. Whether the inflammatory action in these cases was modified by a rheumatic diathesis is uncertain; but in its lighter grade, when characterized merely by pain and stiffness, or impairment of muscular power in the limbs, it was generally reported as chronic rheumatism." He states that injuries may have determined the localization of the inflammatory results of exposure. He says: "Over-exertion and heavy burdens on the loins would predispose by repetition as surely as a more pronounced injury inflicted but once." The number of white soldiers, he points out, that were reported sick with paralysis amounted to 2,837, the deaths to 231, and the discharges to 2,838. He states: ". . . some, reported originally as chronic rheumatism, were discharged on account of aggravation of the spinal affection manifested by paralysis and muscular tremors, and paralytic sequelae of such diseases as the continued fevers also contributed to the total of those discharged. Perfect recovery, implying ability to perform military service, was unusual in these cases of spinal affection. In some of the cases the patients were discharged with the diagnosis of paralysis agitans."

Mott[65] also made observations regarding these paralytic cases. He reported: "A disease much noticed at the present time by that part of the profession connected with the army has been variously named general neuralgia, myalgia, or, from its chief location, intercostal neuralgia." Hyperesthesia was a striking feature. "Its prominent characters," he says, "are the following: Coming on gradually, showing itself first in a portion of the spine, gradually extending around the chest and down the extremities, though in some cases fixed in a particular situation, as in the lower bowels." He observed that the bodies of these patients were bent at an angle, and any effort to assume the upright position was attended with so much pain and distress that the patient would not make the attempt. At his hospital there were no fatal cases. He attributed the disease to malarial emanations from southern swamps.

The symptoms in the above cases suggest both beriberi and poliomyelitis. That these two diseases existed in the United States following the Civil War is indicated by a statement by Allen (1897).[39] He says: "In American books malaria is mentioned as a possible cause of neuritis but it does not seem to be regarded as a common one; possibly a closer study of paralysis of the legs which occurs in malarial regions of the southern states may show that neuritis from this cause is not rare." The early history of poliomyelitis, as reported by Paul,[30] would indicate that epidemics of multiple neuritis (beriberi and poliomyelitis) were also common in the United States prior to the Civil War. He points out, which tends to support this fact, that operations for clubfoot became popular in this country a little further back than 1841.

The following remark by Snyder[66] appears to be pertinent and tends to explain the confusion that has existed in this country regarding poliomyelitis and its related diseases. He says: "The rule governing the history of a disease is that it exists some years among a people before it is known to that people by any particular name and that the disease exists for many years before appearing in the literature of that people.

Multiple neuritis existed in the United States prior to the Civil War. S. Weir Mitchell (1866),[3] in his article, "Paralysis from Peripheral Irritation with Reports of Cases", points out that the writings of many of the older physicians, as well as those of a later day, contain numerous cases of palsy, local or general. "To

such cases," he says, "various names have been affixed, with the intention of grouping a set of unexplained facts, or of indicating by the nomenclature the writer's view of the mode in which the malady was produced. . . . It thus happens that the cases referred to have been classified under such terms as idiopathic paralysis, essential paralysis, functional paralysis, inhibitory palsy, paralysis by exhaustion, reflex paralysis. . . . The industry of modern clinical observers has increased the number of these cases, and it has gradually become apparent that they differ very widely as to their original causation, and perhaps also as to the condition of the neural organs which corresponds to the various groups. *Of late, however, the temptings of an ingenious theory, set forth by a neurologist of great ability, has led many physicians to follow him in a view of these cases which unites them all in a single class, whose nomenclature is meant to point out the pathological mechanism which gives rise to their occurrence.*"

In relating his observations during the Civil War, Mitchell states: "I myself have seen in the wards of the U.S.A. Hospital for Diseases of the Nervous System many cases of palsy, chiefly paraplegia, following upon dysentery, acute or chronic; but in nearly every case there had been many possible causes, such as long marches, bad diet, malaria, or injuries to the spine, these being so common that almost any patient long in service had some such to relate."

A further perusal of the reports that emanated during the Civil War indicate that definite signs of pellagra were also noted and likewise apparently arose from miasmic poisoning. These are noted in the reports concerning the mouth and skin in the malarial diathesis.

Surgeon E. S. Bidwell,[91] 31st Mass. Vols., says: "It is a very peculiar and abnormal appearance of the tongue, in which the under surface appears to have trespassed upon the upper, the papillae of the latter being supplanted by the transverse rugae of the former. The sides are thickened and rounded, the normal well-defined edge being obliterated, and the line of demarcation moved nearer to the mesial line. This appearance of the sides may be associated with any and every appearance of the remaining papillary surface, clean or coated, thick or thin, light or dark, just as the malarious disease may be attended by any and every variety

of morbid condition of the system. Through all this variety it is perfectly distinct, and, when once learned by actual inspection, it is unmistakable. . . . The most fixed condition of this symptom is an appearance of indentation or crimpling, transversely, which is apparently confined to the adjacent tissue, while the superficial tegument is moist, smooth and transparent. In a word, it seems to be a continuation or encroachment of the inferior surface upon the superior and lateral borders of the tongue, greater as we approach the root of the tongue."

Osborn[92] also noted a peculiar appearance of the tongue in malarious diseases and spoke of it as a new variety of malarial fever. He observed that the tongue had a bluish tinge, which is often lost or merged in the various tints or furs produced by other diseases. There was a crenated or crimpled condition of the edges which was not due to the impression of the teeth, for the transverse lines are closer together than would happen if this was the case, and they were observed in infancy and old age when no teeth were present."

Smart[83] states that there were among white troops during the Civil War 32,000 cases of itch, and 35,667 of skin disease. The incidence was 30 per thousand among the whites and 35 among the colored troops. Among the skin conditions, he speaks of papules which became torn and inflamed from itching and then eczematous. He says: "Some western practitioners recognized in it an old acquaintance known by the names of prairie dig, western itch or Missouri mange. . . . Scratching caused the surface to be dotted with dark points of dried blood. Afterward the affected parts assumed an eczematous or even impetiginous character as the result of neglect and violence, and in certain stages of the disease a thin, yellowish, acrid liquid exuded from the inflamed parts. . . . No parasite was found in the exuded fluid. . . . The disease was frequently recorded as itch or camp itch." He emphasizes that, "scabies has no claim to the title of army itch."

Whitehead[93] found the disease indigenous in the southern states, and especially prevalent in Missouri, Arkansas and Tennessee. Radcliffe[34] considered the disease essentially an eczema. He says: "It spreads irregularly over the whole surface of the body, but was found principally on the hands, fingers and elbows, scattered in distinct red spots and vesicles from the axilla to the

wrist, on the nates and particularly in the popliteal space and about the ankles. The skin was frequently so stiff from the crusts and painful from excoriated fissures as, with the occasional itching, to occasion an almost unsupportable nervous action. The vesicles contained a highly excoriating limpid fluid which was capable of extending the diseased action. No parasite was discovered. The disease was attributed to want of cleanliness and continued exposure to the rays of the sun in poorly fed men, and was therefore perhaps of more frequent occurrence among our paroled prisoners than in the ranks of the army."

(*To be concluded in April issue*)

TREATMENT OF INFANTILE PARALYSIS. (Revista Espan. de Pediatria, Zaragoza, 4:133, Jan.-Feb. 1948). The number of cases of poliomyelitis seen in Spain by the author since 1941 is about 1,000. The author found that muscular spasm is independent of paralysis. It is present in all cases of poliomyelitis. Acute muscular spasm is of better prognosis for functional muscular recovery than subacute spasm. Muscular blockage or pseudo-paralysis, alone in antagonistic muscles and in combination with spasm in certain antagonistic muscles, muscular incoordination in either spastic or pseudoparalytic muscles and disturbances of the peripheral structures are frequent. Kenny's treatment is of value. The technic for early and late application of warm compresses, physical therapy, manual correction of muscular deformities and muscular reeducation is described in detail. The treatment, which should be given only by physicians trained in the proper technic, prevents talipes equinus, flexion-abduction of muscles, obliquity of pelvis, scoliosis and certain aponeurotic retractions. Sequels due to destruction of motor neurons are incurable.—*Journal A.M.A.*

A SIMPLIFIED METHOD OF THE CARE OF THE SKIN OF THE NEWBORN*

HARRY APFEL, M.D.
Brooklyn.

This extremely simple method of the care of the newborn baby was first put into operation by us at the Unity Hospital, Brooklyn, New York, in 1927. Since that year our nursery has admitted and discharged over 23,000 newborn babies from our maternity service. As a result of this method we have been spared the costly and unpleasant experience of interrupting the maternity service of our institution so often mandatory in case of an outbreak of some type of skin infection among the babies of the nursery. This utopia was interrupted once when a special nurse disregarded our standing routine and employed her own method in caring for the skin of the infant in her charge at the time. That baby developed a mild case of pyoderma. As a result of prompt and strict isolation no other baby was infected. That one baby also promptly recovered.

In these two decades, many reports have been published on "improved" methods intended to reduce the incidence of skin infections of newborns. All of these methods have one common denominator, namely, some special oil or ointment. Certain pharmaceutical firms have exploited their special kind of "antiseptic" oil which is reputed to have all the virtues of a prophylactic. Indeed, every modern nursery, public as well as private, is well supplied with flasks of some sort of this oil. What a false sense of security for the executive entrusted with the responsibility of the safety and the well-being of the newborn baby against skin infection. Every so often this sense of security is dispelled by an outbreak of an infection, e.g., impetigo or pemphigus. All this adds up to the sum total that the "prophylaxis" does not live up to its reputation. The doctor in charge of a particular nursery is frequently puzzled how to explain the reason for the outbreak of the infection, when he is assured that the personnel did not fail to carry out the particular routine, namely, that the babies receive the special oil or the particular unguent as ordered. Very often, a mother of a newborn will ask us why it is that her baby

*Read in part before the Clinical Society of the Unity Hospital, Brooklyn, New York, March 24, 1948.

developed some form of skin infection in spite of her diligent application of the "antiseptic" oil? Our answer to that mother is always the same, namely, that it is difficult not to injure the baby's skin in the act of massaging it with the oil and, inasmuch as the skin is never quite sterile, the break of the superficial layer of the skin is an invitation for the bacteria to bring about impetigo, etc. We will develop this thought more in detail by citing some of the studies from the literature on this subject.

Potter and Abel[1] report the results of their observations on a routine adopted at the Orange Memorial Hospital, Orange, New Jersey: "Soap and water bath followed by inunction with ammoniate of mercury 3 per cent, soon after the birth of the baby. Every day thereafter the skin is anointed with 'a medicated blended' oil. 1,310 newborn babies were thus treated, 26 developed skin infections of which 12 were impetigo."

Perlstein,[2] in a study at a Chicago hospital (1942-1945), including 2,000 babies, concludes as follows: "The greatest number of skin infections occurred in babies treated with 'antiseptic' oils." "No antiseptic oil will prevent impetigo." This same observer later switched to a "special" lotion, as a result of which, the incidence of skin infections dropped to less than 10 per cent. It is superfluous to ask the reader if that would be satisfactory. I should dread the thought to have to anticipate a 10 per cent incidence of impetigo in a nursery in my charge.

Weymuller and Ittner,[3] in their study at the Long Island College Hospital, covering over 3,000 babies, employed three different routines as prophylactic measures, including corn oil bath, sulfathiazole ointment and ammoniate of mercury. In each of their groups some form of skin infection developed—in one of their groups four cases of pyoderma; in a second group, 35 cases of the same and in the third group, 17 cases of a macul-papular rash.

We could cite many more similar reports from recent publications. Their conclusions are encouraging, namely, a reduced incidence of skin infection. It has always been our aim to eliminate even this reduced incidence. The simple method we wish to report herewith measures up to that objective. We have tested this method for a period of over twenty years, and it has withstood this test without a flaw. This is especially noteworthy in as much as part of that time was the war period when we had

to contend with many shortcomings. Outbreaks of skin infections were prevalent in some of the hospitals of our borough during those years, while our institution was spared this unpleasant experience.

METHOD EMPLOYED AT THE UNITY HOSPITAL

This method is based on the premise that the vernix caseosa is a necessary protective covering which serves as a lubricant to the baby's skin. It should be preserved. After it has served its purpose it will dissolve by normal body temperature without any interference on our part. The baby's skin will be found to be soft, clean and pink. No oil as yet discovered can improve on this task. This spares the break in the superficial layers of the skin which is unavoidable when massaging or anointing with oil is the practice. It has been shown[2] that the skin of the newborn baby is rarely sterile to culture.

The baby is wrapped in soft, warm blankets. The eyes are given the usual prophylactic treatment; the cord is securely tied and wrapped in sterile gauze saturated in 70 per cent alcohol and secured with a narrow band. The baby is soon after dressed in shirt and diaper. These are changed as often as necessary. Any mucus, blood or other discharges are wiped away with clean cotton moistened with warm water. The baby does not get a bath until the cord has come away, which is sometime after the baby has left the hospital or nursery, and not before. The mother is given instructions not to apply or massage any oil on the skin of the baby at any time. She is taught how to continue the routine practiced at the hospital nursery.

CONCLUSION

We have presented our results with the care of the newborn babies at the Unity Hospital nursery—a simple routine of the care of the skin. This method was instituted by us in 1927 and has covered some 23,000 newborns. During that period of time several of the hospitals in our borough have had epidemics of skin infection which made it mandatory for them to suspend maternity service and go through costly "cleaning up" operations and suffer economic losses not to mention the more important child morbidity. Our routine is recommended for a trial in other institutions.

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884 *Park Place.*

LEUKEMIA AND SALMONELLOSIS (S. Dublin). (Nederlandsch Tijdschtift v. Geneeskunde, Amsterdam, 92: 1919, June 26, 1948). Tegelaers and his associates present the care of a 2½-year-old child, daughter of a stock breeder, who had acute lymphatic leukemia and salmonellosis. Bacteriologic examination of the blood revealed Salmonella dublin. The agglutination titer of the patient against this type of organism was 1:1,600. The feces also contained S. dublin. Since S. dublin occurs frequently in cattle, an examination was instituted to ascertain whether the salmonella infection in the child was in any way connected with a germ carrier in the cattle. A cow was found to excrete regularly with the feces a large number of S. dublin organisms. It may be assumed that the salmonellosis in the little patient had arisen secondarily to the leukemia.—*Journal A.M.A.*

BCG VACCINATION. (Prensa Médica Argentina, Buenos Aires, 35:1425, July 23, 1948). Sayago and collaborators report on a large group of infants and children given BCG vaccination and a large group of infants and children who were not given the vaccination. The general mortality within the first year of life reached 8.3 per cent among the vaccinated and 15.6 per cent among the control group. A weak degree of tuberculin sensitivity was prevalent among the vaccinated in contrast to the strongly positive results of Mantoux's test eventually found in children belonging to the control group. Chest roentgen examinations performed on the vaccinated group disclosed 57.5 per cent of lesions among children with tuberculous contact and 18.9 per cent among the noncontacts, while in the control group there were 74.3 per cent of lesions in children exposed and 47.6 per cent in the noncontacts. In addition the roentgenograms in the vaccinated group proved to be less important than the ones seen in the nonvaccinated children, especially in the case of primary infection when the sole lymph node enlargement could be detected in the former.—*Journal A.M.A.*

CLINICAL REVIEWS

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York; Doctor Philip Moen Stimson, Cornell Medical School, New York, and Doctor John A. Toomey, Western Reserve University Medical School, Cleveland. Other interested medical schools are cordially invited to submit student papers for consideration.*

THROMBOCYTOPENIC PURPURA COMPLICATING RUBELLA*

REPORT OF CASE

MYRON FREEDMAN, M.D. and PETER LADERMAN, M.D.
New York.

Although rubella is usually considered to be one of the mildest of the childhood diseases, it is occasionally followed by complications, one of the rarest of which is thrombocytopenic purpura. Fox and Walton,[1] who in 1946 described two well authenticated cases, state that a review of the literature up to that time revealed only two previously reported instances in which thrombocytopenic purpura complicated rubella. However, five rather poorly substantiated cases had been reported between 1871 and 1911. Magnusson[2] and Warren, Rogliand and Potsubay[3] reported a total of four more cases in 1946. In addition, Magnusson states that Rudisill,[4] describing the roegnten ray treatment of thrombocytopenic purpura, mentions in a footnote that the case thus treated occurred in a 23-year-old girl following rubella. This sequence has been claimed to occur, therefore, in a total of 14 previously reported cases, and has been well substantiated in eight or perhaps nine instances. In addition, thrombocytopenia has been reported complicating scarlet fever, varicella,[8] measles,[9], infectious mononucleosis[10] and upper respiratory infections. The case described below is of special interest because it occurred in one of two apparently identical, pseudohermaphroditic female twins. Although both children suffered from rubella at the same time, only one displayed any hemorrhagic tendencies.

*Submitted as partial fulfillment of the requirements of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals.

CASE REPORT

L. T., a three-year-old Puerto Rican female, born November 29, 1942 at Metropolitan Hospital, was admitted to the pediatric ward of Flower and Fifth Avenue Hospitals on June 5, 1946, with a chief complaint of many purplish spots over the arms, legs and abdomen. The patient and her sister, apparently identical, pseudohermaphroditic twins, had recovered from rubella, which had been diagnosed by a private physician, about one week prior to admission. The child had been admitted to Flower Hospital twice previously for investigation of the true sex of both children. At birth there was a single placenta and investigators were reasonably sure that the twins were identical. The child in question was first admitted from February 18 to March 2, 1946, at which time she developed measles and had to be transferred to Willard Parker Hospital. She was readmitted to Flower Hospital on March 8, 1946 and subjected to exploratory laparotomy, which revealed a normal uterus, Fallopian tubes and ovaries in their normal anatomical position. A patent vagina was discovered, in apparently normal relationship to the cervix uteri. Recovery from the surgery was uneventful. The child's past history included frequent colds, enlarged tonsils and frequent constipation. Two or three months prior to admission in June 1946, she had had chickenpox, followed by a yellow discharge from the left ear which lasted a month.

Physical Examination at the time of the third admission revealed the following abnormalities: 1. A few petechial hemorrhages over the pharyngeal wall. 2. Purpuric areas, varying in size, diffusely distributed over the abdomen, legs and arms. 3. Mild cervical lymphadenopathy. 4. Bilateral hypertrophic tonsils. 5. A mucopurulent nasal discharge. 6. A questionably palpable spleen. 7. A positive Rumpel-Leede capillary fragility test was obtained.

Laboratory Results were as follows: red blood count, 4,850,000 cells per cubic millimeter; hemoglobin, 10.8 grams per 100 cc. of blood (70 per cent); color index, 0.7; white blood count, 10,500 cells per cubic millimeter with 71 per cent polymorphonuclears, 20 per cent lymphocytes, 8 per cent mononuclears and 1 per cent Turk cells. There were 36,000 platelets per cubic millimeter on admission, and the prothrombin time was 19.1 seconds. In the vitamin C skin test, the dye was absorbed in 27 minutes 10 seconds,

and the plasma ascorbic acid was 1.12 milligrams per 100 cc. of blood. The latter two tests were interpreted as showing some vitamin C deficiency. Routine and microscopic urinalyses were negative, and the blood Wassermann was negative.

Diagnosis, based on clinical findings and laboratory examination, was thrombocytopenic purpura secondary to rubella. The child was treated symptomatically for 9 days until June 14, 1946, when there was noted marked recession of her old purpuric spots without the appearance of any new hemorrhages. On June 10, the platelet count was 65,000 per cubic millimeter. From June 14 to June 17, the child's temperature was elevated, reaching 104° F. at times. Both tonsils were injected and covered by exudate. A diagnosis of acute follicular tonsillitis was made, and 10,000 units of penicillin were administered intramuscularly every three hours with a prompt drop in temperature and improvement of the patient's symptoms. The penicillin was discontinued on June 19. From June 17 until the day of discharge, 200 milligrams a day of ascorbic acid, 15 drops of oleum percomorph, and 5 drops of Blexin were given. The platelet count on June 18 was 140,000 per cubic millimeter. A complete blood count done on June 24 showed the following: red blood count, 4,500,000 per cubic millimeter; hemoglobin, 70 per cent; color index, 0.8; white blood count, 6,800 with 46 per cent polymorphonuclears, 53 per cent lymphocytes and 1 per cent mononuclear cells; platelets, 220,000. The vitamin C skin test showed absorption of the dye in 6 minutes 10 seconds, indicating a normal ascorbic acid blood level.

By June 25, the purpuric eruption had completely disappeared and the platelet count was normal. The patient was discharged on June 27 with the following diagnoses:

1. Thrombocytopenic purpura secondary to rubella.
2. Pseudohermaphroditism.
3. Acute follicular tonsillitis.
4. Vitamin C deficiency.

DISCUSSION

Although this patient was not hospitalized during the active phase of her rubella, the history is probably accurate, especially since the child was known to have had true measles during a previous admission, the chief disease which must be differentiated from

rubella. Posterior cervical lymphadenopathy was present at the time of admission. The diagnosis of thrombocytopenic purpura was well borne out by clinical and laboratory evidence, so that this case may be added to the brief list of instances in which thrombocytopenic purpura has complicated rubella. Fox and Walton[1] quote Quick[11] as listing the following criteria for the diagnosis of thrombocytopenic purpura: 1. Capillary hemorrhage, usual cutaneous or mucosal. 2. Prolonged bleeding time. 3. Platelet count below 100,000, and usually below 60,000. Adjuvant signs are: normal or slightly elevated clotting time, positive Rumpel-Leede test, delayed clot retraction, normal or slightly elevated leukocyte count (to differentiate this purpura from that of granulocytopenic leukemia or aplastic anemia). In the case reported here, two of the three major criteria were fulfilled, the bleeding time not having been performed. The Rumpel-Leede test was positive, and the leukocyte count was somewhat elevated. Clotting time and clot retraction tests were not done.

The previously reported cases and the one herein described have been similar in nature. The purpura has followed the onset of rubella by a period of from 4 to 10 days. The bleeding may first become evident from either the skin or the mucous membranes, but in most cases both are eventually involved, and rarely there is evidence of cerebral bleeding, melena or hematuria.[3] The distribution of the lesions has been widespread, and they have remained evident for a period of time varying between days and months. Splenic enlargement has not been a frequent finding. In no case has a personal or family history of bleeding tendencies been obtained. The case described here is especially interesting in that respect, since identical twins, very similar in hereditary constitution, environment and diet, had rubella at the same time; yet only one displayed hemorrhagic tendencies.

Quick's three criteria were fulfilled in all cases in which the tests involved were reported, and the minor criteria have usually been satisfied, too. In all cases reported to date, there has been complete recovery from the purpuric complication, which has usually been mild. But the two cases which occurred in American soldiers, reported by Warren and his co-workers,[3] had hematuria, and one case had melena. The urine in one of these cases looked like gross blood and contained 1,150,000 red cells per cubic milli-

meter. There were marked bladder symptoms. Six transfusions of fresh, citrated whole blood failed to stop the bleeding immediately.

Most cases require only symptomatic therapy, but Warren, Rogliand and Potsubay have advised the replacement of lost blood by transfusions of fresh, citrated whole blood and the application of styptics to accessible bleeding areas.

The etiological connection between rubella and purpura is not clear. Fox and Walton state that the two important factors in the production of any purpura are: 1. increased capillary permeability; and 2. decreased platelet count.[1] These authors further state that increased permeability of capillaries, due to vasodilation and not reduction in the number of platelets, seems to be the more important factor in producing the purpura which is associated with rubella. This contention is partly substantiated by the experience of Warren and his co-workers,[3] who found that the purpura receded following transfusions of fresh, citrated whole blood, even though the platelet count remained about 50,000.

Magnusson,[2] too, attributes the purpura to a change in capillary resistance, perhaps due to an allergic reaction to the rubella virus, and discounts the role of thrombocytopenia. He points to the widespread distribution of the petechiae as evidence for a capillary weakness as a basis for the purpura, stating that the petechiae found in purely thrombocytopenic purpura are more limited in distribution. He describes the purpura as being "dys-vascularly-allergic." The six and eight day intervals, respectively, between the onset of rubella and the first appearance of hemorrhagic tendencies in his two cases he believes also lend support to an allergic basis for the purpura. Kernau,[7] cited by Magnusson, has discovered that a very slight lowering of capillary resistance is a part of rubella, but this would not seem to account for the widespread and sometimes severe purpura which is seen. Hemorrhagic rubella, probably due to this phenomenon, does occur rarely, but when it does, the hemorrhages are of a different nature, milder, and not accompanied by thrombocytopenia.

Although the patient reported in the present case was shown to have a vitamin C deficiency, there is no necessary connection between the deficiency and the purpura, since poor nutrition is characteristic of the national and economic group to which this

child belongs. A more definite statement as to the role of the vitamin deficiency could be made if ascorbic acid levels had been done simultaneously on the twin sister, who presumably ate the same food, and if we knew more of the dietary habits of these two children. In addition, the plasma vitamin C level was higher than the skin test time would seem to indicate. The history reveals, moreover, that the purpura had begun to recede prior to the commencement of supplementary vitamin therapy, although it might be supposed that the ordinary hospital diet would suffice to correct a mild deficiency. Fox and Walton[1] do not believe that vitamin deficiency plays an important role in the etiology of this disease.

CONCLUSIONS

Rubella, usually a mild childhood illness, may sometimes be followed by a thrombocytopenic purpura of an alarming nature, but all cases so far reported have recovered. This complication has occurred in patients who have not previously displayed hemorrhagic tendencies, and in whose family history there is no indication of a hereditary bleeding tendency. The etiology is not clear, but decreased capillary resistance, perhaps as an allergy reaction to the disease-producing organism, seems to be the chief factor, although the platelet count has been lowered in all cases in which this examination was performed.

Usually no specific therapy is necessary, but repeated transfusions of fresh, citrated whole blood, and the local application of styptics may be helpful in severe cases.

SUMMARY

1. The literature pertaining to thrombocytopenic purpura complicating rubella has been reviewed.

2. A new case has been reported.

3. The characteristics of the syndrome have been summarized.

4. Hypotheses relating to the etiology of thrombocytopenic purpura complicating rubella have been discussed.

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CURARE TREATMENT OF POLIOMYELITIS. (Nordisk Medicin, Stockholm, 37: 476, March 5, 1948). Normann says that curare, tried in 6 in a later stage, was of no definite value in the acute phase but gave good results in 3 of the 4 cases with beginning contractures. Side effects in some instances indicated termination of the treatment. The dosage should be individualized because of the danger of overdosage, as in cases with involvement of the respiratory tract. No conclusion could be drawn as to the efficacy of curare in combination with more radical physical therapy.—*Journal A.M.A.*

STREPTOMYCIN IN TUBERCULOUS MENINGITIS. (Semana Médica, Buenos Aires, 55: 1057, June 24, 1948). Gallo reports 2 girls aged 16 and 26 years, respectively, with tuberculous meningitis. Streptomycin was given by intraspinal and intramucular injections. The younger patient received a total dose of 68 Gm. intrathecally and 78 Gm. intramuscularly. Clinical cure was attained which has persisted more than one year after discontinuation of treatment with streptomycin. There are no pulmonary lesions. The older patient received a total dose of 64 Gm. intramuscularly and 3 Gm. intrathecally. Definite improvement followed, which lasted for three months after discontinuation of streptomycin therapy. Acute symptoms including symptoms of involvement of the central nervous system appeared suddenly and terminated fatally. Necropsy revealed acute cerebral lesions. The author concludes that streptomycin is the only hope of cure for patients with tuberculous meningitis, the prognosis of which was fatal before discovery of streptomycin and is now slightly but favorably modified.—*Journal A.M.A.*

BOOK REVIEWS

PSYCHODYNAMICS AND THE ALLERGIC PATIENT. By Harold A. Abramson, M.D., with a Panel Discussion. Cloth. Pp. 81. Illustrated. Price $2.50. St. Paul and Minneapolis: Bruce Publishing Co., 1948.

This brief volume contains a rather pleasant discussion of the importance of the psyche vis a vis the soma, especially as it relates to allergic conditions. The discussion was sponsored by the American College of Allergists at its Third Annual Meeting which was held in Atlantic City, N. J., on June 8, 1947. The tenor of the majority of the discussers is that the patient must be considered as a whole, considering always the effect of the psyche on whatever somatic condition that might be present. Although the subject was limited to allergy, this principle can be extended to all fields of medicine. As one of the speakers said, "the purpose of the discussion is not to study the soma less but to study the psyche more."

MICHAEL A. BRESCIA, M.D.

PRÉCIS DE MÉDICINE INFANTILE. By Clément Launay, M.D. Paper. Price 1150 fr. Pp. 1018. Illustrated. Masson & Cie., 120, boulevard Saint-Germain, Paris, 6e, 1948.

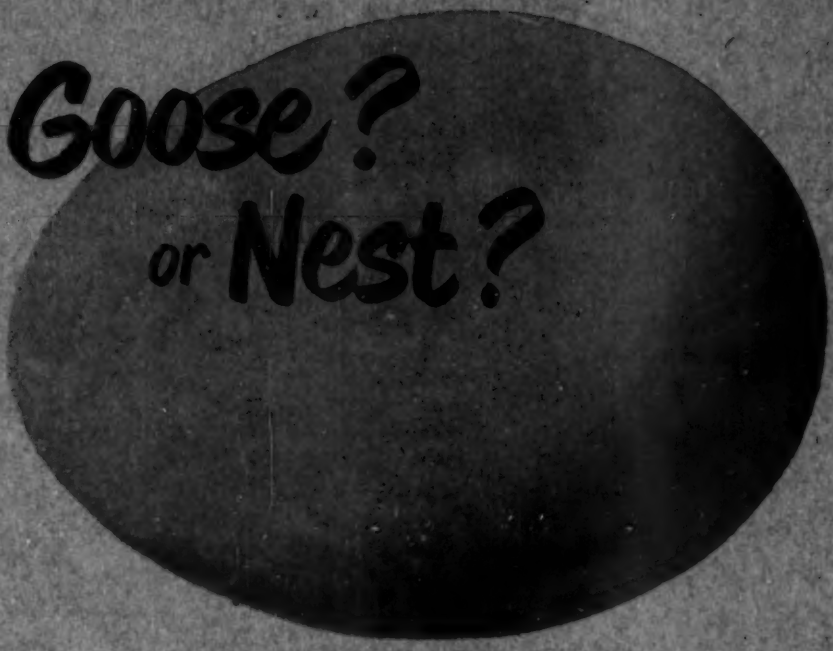
This is a fairly comprehensive book of pediatrics. For the most part it can be used as a reference text for a brief description of the many diseases which are characteristic of the infant and young child. The book is up-to-date with few exceptions. The treatments for pertussis and acute poliomyelitis are inadequate, and immunization against diphtheria is advised at one year of age which at present is considered too late.

MICHAEL A. BRESCIA, M.D.

PREMATURE INFANTS. A MANUAL FOR PHYSICIANS. By Ethel C. Dunham, M.D. Paper. Price $1.25. Pp. 401. Illustrated. Washington, D.C.: Superintendent of Documents, U.S. Government Printing Office.

This book is for all practical purposes a review of the literature as it pertains to the premature infant. The author has inserted a concise summary at the end of each chapter heading. The book contains a wealth of information and is replete with statistics for those who are interested. This volume is difficult to read because of the format but nevertheless it has some value as a reference work.

MICHAEL A. BRESCIA, M.D.



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Every growing child needs a diet balanced as to calorie, protein and vitamin requirements. But, often, a theoretically correct diet may actually be vitamin deficient. For instance, the child may eliminate habitually certain foods; or digestive disturbances may interfere with assimilation. Improper or excessive cooking also will dissipate certain vitamins. A continued mild vitamin deficiency can result in fatigability, irritability, skin eruptions, susceptibility to colds and infections.

DABS Armour (improved formulae)

the balanced vitamin capsules, supply needed supplementary vitamin administration. DABS contain a vitamin mixture similar to that contained in a high-grade vitamin-rich diet, plus liver extract.

Each DABS (improved formulae) capsule contains:

Vitamin A	10,000 U.S.P. Units (2½ M.D.R.)
Vitamin D	1,000 U.S.P. Units (2½ M.D.R.)
Vitamin B_1	5.0 Mg. (5 M.D.R.)
Vitamin B_2	10.0 Mg. (5 M.D.R.)
Vitamin C	75.0 Mg. (2½ M.D.R.)
Niacin Amide	50.0 Mg. (M.D.R. Not Estab.)
*Pyridoxine HCl (Vit. B_6)	0.25 Mg.
*Calcium Pantothenate	5.0 Mg.
Secondary Liver Fraction	0.5 Gram

M.D.R.—Minimum Daily Requirement.
*Need in human nutrition not yet established.

ARMOUR Laboratories

Have confidence in the preparation you prescribe—specify "Armour."

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